

patients (20%) and normal in 105. All 8 of the patients with minor elevations of CK-MB also had elevated troponin T; CK-MB was normal in all patients with normal troponin T. Patients with elevated troponin T levels were older (74 ± 10 vs 63 ± 12 years, $p < 0.001$) but did not differ from patients with normal levels with respect to risk factors or other clinical features. However, as shown in the table, coronary events occurred significantly more frequently in patients with elevated troponin T levels:

Troponin T on admission:	Normal	Elevated	p
Patients	105	26	—
Myocardial infarction	3 (3%)	8 (31%)	<0.001
Refractory angina	9 (9%)	4 (15%)	NS
Revascularization	32 (30%)	17 (65%)	=0.001
Pts with cardiac events	42 (40%)	25 (96%)	<0.001

Only one of the 26 patients with elevated troponin T levels was controlled with medical therapy alone, compared to 62 of the 105 with normal levels (4% vs 59%, $p < 0.001$). The cutpoint of $>0.1 \mu\text{g/L}$ had a sensitivity of 50% and a specificity of 87% in predicting refractory angina/myocardial infarction.

Conclusion: Serum troponin T levels are elevated on admission in one fifth of unselected unstable angina patients. These patients have a very high rate of myocardial infarction and refractory angina despite medical treatment, and most of them undergo revascularization. Troponin T levels are an independent and potentially useful predictor of early coronary events in unstable angina.

9:30

737-5 Risk Stratification in Acute Ischemic Syndromes Using Serum Troponin T

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To evaluate whether serum Troponin T (Trop-T) or CK-MB analysis could be used to risk stratify patients with acute ischemic syndromes, admission serum samples were obtained in 865 patients enrolled in the GUSTO-IIa study. Pts were stratified on admission into ST elevation ($n = 531$) and non-ST elevation ($n = 334$) groups. CK-MB (mass assay) and Trop-T (immunoassay) were measured by an enzymatic core laboratory. The median (25th, 75th percentile) serum CK-MB was 2.9 (0.9, 8.2) ng/ml in the non-ST and 4.0 (1.5, 14.1) ng/ml in the ST elevation group. Corresponding Trop-T levels were 0.05 (0.01, 0.27) ng/ml and 0.04 (0.01, 0.33) ng/ml, respectively. Elevated levels of CK-MB (>7 ng/ml) were seen in 33% and Trop-T levels >0.1 ng/ml were seen in 36% pts. Regression analysis documented that Trop-T levels were strongly associated with mortality ($p < 0.0001$), while CK-MB levels were less strongly associated ($p = 0.03$). In a regression model of both serum markers and mortality, CK-MB added no information ($p = 0.9$) after the Trop-T levels were included. Higher admission Trop-T levels were also associated ($p = 0.005$) with a poor clinical outcome using a composite endpoint of death, late (>18 hours after admission) myocardial infarction/reinfarction (MI), cardiogenic shock or heart failure (CHF), while only a weak relationship was observed with CK-MB ($p = 0.07$). Outcomes based on admission Trop-T levels >0.1 ng/ml (Pos) or <0.1 (Neg) were as shown:

	Non-ST Group		ST elevation Group	
	Trop-T Neg	Trop-T Pos	Trop-T Neg	Trop-T Pos
Outcomes	N = 201	N = 123	N = 324	N = 176
Death	1%	9%	5%	12%
Shock	2%	6%	3%	9%
MI	6%	11%	5%	7%
CHF	7%	16%	10%	15%

In conclusion, Troponin T measurements in patients with acute ischemic syndromes can stratify patients into high and low risk categories. Early and rapid determination of Troponin T levels has the potential to streamline health care delivery in patients with acute ischemic syndromes.

9:45

737-6 Combining Myoglobin and Clinical Variables for Assessing Coronary Reperfusion After Thrombolytic Therapy

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Myoglobin (MYO), measured using a quantitative 10 minute assay, was examined in combination with clinical variables for assessing infarct related artery (IRA) patency in 96 MI pts, all of whom received front-loaded throm-

bolytic therapy (TT) at a median (25th, 75th percentile) of 162 (113, 226) mins after onset of symptoms. All pts had acute angiography (Cath) at 142 (96, 195) mins after TT; flow in the IRA was graded as either "OPEN", TIMI 2-3 ($n = 71$), or "CLOSED", TIMI 0-1 ($n = 25$). MYO was determined in serial samples collected before TT (Baseline) and within 10 mins of Cath. MYO values, in ng/mL, in Baseline samples were similar for the OPEN and CLOSED groups at 112 (51, 275) and 81 (47, 195), respectively; for samples collected at Cath, values were 915 (501, 2237) for the OPEN group and 273 (159, 855) for the CLOSED group. MYO strategies based on: 1. MYO Value at Cath, 2. change from baseline to Cath (Delta), and 3. rate of release (slope) were each combined with clinical variables for discriminating OPEN from CLOSED pts. The clinical variables examined were: time from Chest Pain (CP) to TT, time from TT to Cath, pt weight, pt age, MI location, race, and intensity of CP at Cath graded 0-10. By logistic regression, the best model for discriminating OPEN from CLOSED pts resulted from combining the following:

Strategy	χ^2	p-value
MYO value at Cath	17.97	0.0002
CP at Cath (0-10)	7.46	0.006
Combined Model	23.95	>0.000001

This combined model yielded a C-Index of 0.82, indicating that the strategy could be clinically useful for discriminating between the OPEN from CLOSED pt groups. This combined strategy is convenient for noninvasively assessing reperfusion status after TT because it involves a single MYO measurement and a straightforward clinical indicator.

738 Medicine, Angioplasty or Operation

Tuesday, March 21, 1995, 8:30 a.m.-10:00 a.m.
Ernest N. Morial Convention Center, Room 102

8:30

738-1 Five Year Trends in the Treatment of Angiographically Documented Coronary Artery Disease: Experience of a Tertiary Care Center

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To determine the impact of new technology on the management of patients with significant ($>70\%$ stenosis) coronary artery disease (CAD), we compared treatment strategies in patients undergoing diagnostic cardiac catheterization during the years 1987-1989 (group 1) and 1992-1994 (group 2). Patients with significant congenital and valvular disease, left main disease, and insignificant coronary artery disease were excluded from analysis. Although there was a higher prevalence of hypertension (60.6% vs 66.6%, $p = 0.024$), hypercholesterolemia (32.5% vs 51.5%, $p = 0.001$), and female gender (27% vs 33%, $p < 0.001$), and a lower prevalence of unstable angina at the time of the procedure (26.8% vs 17.9%, $p = 0.001$) in group 2, there were no differences between the two groups in mean age (61.0 yrs vs 62.5 yrs), incidence of diabetes (26.5% vs 25.2%), tobacco use (58.4% vs 56.1%), peripheral vascular disease (16.3% vs 17.3%) or cerebrovascular disease (7.8% vs 6.8%). Although there were more female patients in group 2, the percent of women and men undergoing revascularization was similar. Treatment strategies were classified as either medical therapy; percutaneous revascularization; or coronary artery bypass surgery (CABG).

	Group 1 [1987-1989] n (%)	Group 2 [1992-1994] n (%)	p value
Medical Therapy	573 (38%)	808 (49%)	0.001
Percutaneous Revascularization	453 (30%)	472 (29%)	NS
CABG	490 (32%)	370 (22%)	0.001

Despite technologic advances in percutaneous revascularization, the percentage of patients undergoing percutaneous revascularization has not increased. There has been a significant reduction in the percentage of patients referred for CABG, which may be due to an increasing number of patients being treated medically. This change in treatment strategy may reflect better medical therapy and/or increased concern about the risks of revascularization.